

Product Data Sheet

Product Name: Prednisone

Cat. No.: GC16450

Chemical Properties

Cas. No. 53-03-2

Chemical Name (8S,9S,10R,13S,14S,17R)-17-hydroxy-17-(2-hydroxyacetyl)-10,13-dimethyl-6,7,8,9,12,14,15,16-octahydrocyclopenta[a]phenanthrene-3,11-dione

SMILES CC12CC(=O)C3C(C1CCC2(C(=O)CO)O)CCC4=CC(=O)C=CC34C

Formula $C_{21}H_{26}O_5$

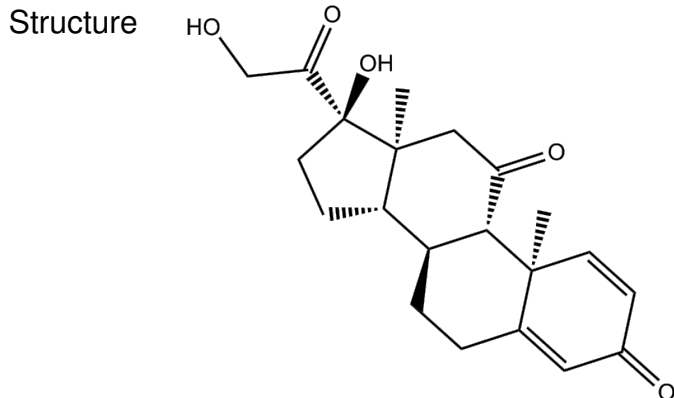
M.Wt 358.43

Solubility $\geq 15.35\text{mg/mL}$ in DMSO

Storage Store at 4°C

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT, or blue ice upon request.



Protocol

Cell experiment [1]:

Cell lines Peripheral blood lymphocytes (PBL) or peripheral CD4+ and CD8+ T cell subsets

Caution: Product has not been fully validated for medical applications. For research use only.

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com

Address: 10292 Central Ave. #205, Montclair, CA, USA

Product Data Sheet

Preparation method	The solubility of this compound in DMSO is > 15.4 mg/mL. General tips for obtaining a higher concentration: Please warm the tube at 37 °C for 10 minutes and/or shake it in the ultrasonic bath for a while. Stock solution can be stored below - 20 °C for several months.
Reacting condition	10-3 ~ 10-12 M; 72, 96 and 120 hrs
Applications	Prednisone arrested PBL in the G1 phase of cell cycle, and inhibited IL-2 receptor (IL-2R) expression as well as IL-2 secretion. In phytohaemagglutinin (PHA)-activated human PBL, Prednisone significantly increased apoptosis. Moreover, Prednisone exhibited stronger apoptotic effect on CD8+ T lymphocytes than on CD4+ T lymphocytes. All these effects were shown in dose- and time-dependent manners.
Animal experiment [2]:	
Animal models	Male Wistar rats
Dosage form	5 mg/kg/d; p.o.; for 90 days
Applications	In Wistar rats, Prednisone significantly delayed learning and memory retention by 20%. Compared with the control group, the neuronal degeneration index in the Prednisone treatment group was 2 times higher in the prefrontal cortex, and about 10 times higher in the CA1 hippocampus. In addition, Prednisone substantially increased the number and cytoplasmic transformation of astrocytes, as well as isolectin-B4-labeled microglia cells in the prefrontal cortex.

Caution: Product has not been fully validated for medical applications. For research use only.

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com

Address: 10292 Central Ave. #205, Montclair, CA, USA

Product Data Sheet

Other notes Please test the solubility of all compounds indoor, and the actual solubility may slightly differ with the theoretical value. This is caused by an experimental system error and it is normal.

References:

- [1]. Lanza L, Scudeletti M, Puppo F, Bosco O, Peirano L, Filaci G, Fecarotta E, Vidali G, Indiveri F. Prednisone increases apoptosis in in vitro activated human peripheral blood T lymphocytes. Clin Exp Immunol. 1996 Mar;103(3):482-90.
- [2]. Ramos-Remus C, González-Castaeda RE, González-Perez O, Luquin S, García-Estrada J. Prednisone induces cognitive dysfunction, neuronal degeneration, and reactive gliosis in rats. J Investig Med. 2002 Nov;50(6):458-64.

Background

Prednisone (Adasone) is a synthetic corticosteroid agent that is particularly effective as an immunosuppressant compound. Target: Others Prednisone is a synthetic corticosteroid drug that is particularly effective as an immunosuppressant drug. It is used to treat certain inflammatory diseases (such as moderate allergic reactions) and (at higher doses) some types of cancer, but has significant adverse effects. Because it suppresses the immune system, it leaves patients more susceptible to infections. Prednisone can also be used in the treatment of decompensated heart failure to potentiate renal responsiveness to diuretics, especially in heart failure patients with refractory diuretic resistance with large dose of loop diuretics. The mechanism is prednisone, as a glucocorticoid, can improve renal responsiveness to atrial natriuretic peptide by increasing the density of natriuretic peptide receptor type A in the renal inner medullary collecting duct, inducing a potent diuresis.

References:

- [1]. RIEMER AD. Application of the newer corticosteroids to augment diuresis in congestive heart failure. Am J Cardiol. 1958 Apr;1(4):488-96.
- [2]. Zhang H, et al. Prednisone adding to usual care treatment for refractory decompensated congestive heart failure. Int Heart J. 2008 Sep;49(5):587-95.

Caution: Product has not been fully validated for medical applications. For research use only.

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com

Address: 10292 Central Ave. #205, Montclair, CA, USA